| L Number | Hits | Search Text | DB | Time stamp |
|---|---|---|---|---|
| 1 | 132 | transgene and (e1a with e1b) | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:43 |
| 2 | 127 | transgene and (e1a with e1b) and promoter | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:43 |
| 3 | 316 | (e1a with e1b) and promoter | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:43 |
| 4 | 127 | (e1a with e1b) and promoter and transgene | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:43 |
| 5 | 520 | e1a and e1b | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:44 |
| 6 | 95 | (e1a and e1b) with vector | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:44 |
| 7 | 37 | ( (e1a with e1b) and promoter and transgene) and ((e1a and e1b) with vector) | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:44 |
| - | 0 | (AAV and adenovir?) with hybrid | USPAT; IBM TDB | 2001/01/31 09:34 |
| - | 113 | (host adj cell?) and rep and cap | USPAT; IBM TDB | 2001/01/31 09:36 |
| - | 0 | ((host adj cell?) and rep and cap) and adeno? | USPAT; IBM TDB | 2001/01/31 09:36 |
| - | 7 | ((host adj cell?) and rep and cap) and ((induc? or comstitutiv?) with promoter?) | USPAT; IBM TDB | 2001/01/31 09:41 |
| - | 15 | ((host adj cell?) and rep and cap) and E1a and E1b and E2a | USPAT; IBM TDB | 2001/01/31 09:54 |
| - | 0 | ((host adj cell?) and rep and cap) and E1a and E1b and E2a | EPO; JPO; DERWENT | 2001/01/31 09:54 |
| - | 254 | helper with virus | EPO; JPO; DERWENT | 2001/01/31 09:55 |
| - | 1268 | helper with virus | USPAT; IBM TDB | 2001/01/31 09:56 |
| - | 0 | (((host adj cell?) and rep and cap) and E1a and E1b and E2a) not (helper with virus) | USPAT; IBM TDB | 2001/01/31 09:56 |
| - | 741 | helper adj virus | USPAT; IBM TDB | 2001/01/31 09:56 |
| - | 0 | (((host adj cell?) and rep and cap) and E1a and E1b and E2a) not (helper adj virus) | USPAT; IBM TDB | 2001/01/31 09:57 |
| - | 3 | (((host adj cell?) and rep and cap) and E1a and E1b and E2a) and transgene? | USPAT; IBM TDB | 2001/01/31 09:58 |
| - | 0 | (((host adj cell?) and rep and cap) and E1a and E1b and E2a) and transgene? | EPO; JPO; DERWENT | 2001/01/31 09:58 |
| - | 15 | (((host adj cell?) and rep and cap) and E1a and E1b and E2a) and (helper with virus) | USPAT; IBM TDB | 2001/01/31 09:59 |

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|---|---|---|---|---|
| - | 3 | 9534670.pn. | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 14:01 |
| - | 3 | 9615777.pn. | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 14:01 |
| - | 1 | 6258595.pn. | USPAT; IBM TDB | 2001/11/16 17:14 |
| - | 70 | E1a adj promoter | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:14 |
| - | 20 | E2a adj promoter | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:14 |
| - | 27 | E1b adj promoter | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:15 |
| - | 1 | (E1a adj promoter) and (E2a adj promoter) and (E1b adj promoter) | USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB | 2001/11/16 17:22 |

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     FILE 'MEDLINE' ENTERED AT 16:59:07 ON 16 NOV 2001
L1            49 S E1A(W)PROMOTER
L2            43 S E2A(W)PROMOTER
L3             1 S E2B(W)PROMOTER
L4            34 S E1B WITH PROMOTER
L5             0 S L4 AND L1
L6            29 S E1A AND E1B AND E2A
L7         84905 S PROMOTER?
L8         16156 S L7 AND 6
L9             9 S L7 AND L6
L10           27 S E1B(W)PROMOTER
L11            0 S L1 AND L10 AND L2
L12            0 S L1 AND L10
L13            0 S L1 AND L2
L14            0 S L10 AND L2
L15            1 S HYBRID AAV AND ADENOVIRUS
L16            0 S TRANSGENE AND REP AND CAP AND E1A AND E2A
L17          131 S TRANSGENE AND AAV
L18           61 S REP AND CAP
L19            7 S L17 AND L18
L20           19 S TRANSGENE AND ITR
L21            1 S L19 AND L20
L22           29 S E1A AND E1B AND E2A
L23      1273466 S INDUC?
L24        84905 S PROMOTER?
L25            1 S L22 AND L23 AND L24
L26           17 S E1A AND CMV
L27            3 S E2A AND CMV
L28            0 S L26 AND L27
L29            3 S HYBRID ADENOVIRUS AND AAV
L30           30 S E2A AND TRANSGENE
L31          453 S E1A AND E1B
L32            0 S L30 AND L31
L33            0 S L30 AND ITR
                 E GAO/AU
                 E GAO G/AU
L34           75 S E3
                 E GAO GUANGPING/AU
                 E GAO GUANGPING/AU
L35            2 S HYBRID AND L34
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L18 ANSWER 7 OF 11 MEDLINE
ACCESSION NUMBER: 95134094 MEDLINE
DOCUMENT NUMBER: 95134094 PubMed ID: 7832644
TITLE: Unusual splice sites in the **E1A-E1B** cotranscripts synthesized in adenovirus type 40-infected A549 cells.
AUTHOR: Ishida S; Fujinaga Y; Fujinaga K; Sakamoto N; Hashimoto S
CORPORATE SOURCE: Cancer Research Institute, Sapporo Medical College, Japan.
SOURCE: ARCHIVES OF VIROLOGY, (1994) 139 (3-4) 389-402.
Journal code: 8L7; 7506870. ISSN: 0304-8608.
PUB. COUNTRY: Austria
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950307
Last Updated on STN: 19970203
Entered Medline: 19950222

AB The adenovirus E1 DNA region consists of two transcription units, E1A and E1B. In this paper we report that the **E1A-E1B** cotranscripts containing sequences of both the E1A and E1B regions are synthesized during adenovirus type 40 (Ad40) infection of A549 cells. Cytoplasmic RNA was isolated from Ad40-infected A549 cells at 24, 72, and 100 h post infection (p.i.). The complementary (c) DNA was synthesized by reverse transcription using an oligo-dT primer and then amplified by the polymerase chain reaction (PCR) using primers derived from the E1A and E1B regions. The cDNAs thus amplified were sequenced either directly or after cloning into bacteriophage M13 **vectors**. Analysis of cDNA indicated that the **E1A-E1B** cotranscripts are synthesized at 72 h p.i., but not at 24 or 100 h p.i. Nucleotide sequences of three cDNAs of the **E1A-E1B** cotranscripts indicated that the cotranscripts originate from the E1A promoter and lack sequences for both the E1A poly(A) site and E1B cap site. The splices create open reading frames for **E1A-E1B** fused polypeptides around the **E1A-E1B** junctions in these mRNAs. Most interestingly, the sequence analysis showed that the 5' and 3' splice junctions in the two **E1A-E1B** cotranscripts do not conform to the splice consensus GT-AG rule. Our results thus suggest that factor(s) which lead to unusual splicing in the E1 mRNAs are present in Ad40-infected A549 cells.

L18 ANSWER 3 OF 11 MEDLINE
ACCESSION NUMBER: 1998105729 MEDLINE
DOCUMENT NUMBER: 98105729 PubMed ID: 9444984
TITLE: Production and characterization of improved adenovirus **vectors** with the E1, E2b, and E3 genes deleted.
AUTHOR: Amalfitano A; Hauser M A; Hu H; Serra D; Begy C R; Chamberlain J S
CORPORATE SOURCE: Department of Pediatrics, Duke University Medical Center, Durham, North Carolina 27710, USA.. amalf001@mc.duke.edu
SOURCE: JOURNAL OF VIROLOGY, (1998 Feb) 72 (2) 926-33. Journal code: KCV; 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980226
Last Updated on STN: 19980226
Entered Medline: 19980218

AB Adenovirus (Ad)-based **vectors** have great potential for use in the gene therapy of multiple diseases, both genetic and nongenetic. While capable of transducing both dividing and quiescent cells efficiently, Ad **vectors** have been limited by a number of problems. Most Ad **vectors** are engineered such that a transgene replaces the Ad **E1a**, **E1b**, and E3 genes; subsequently the replication-defective **vector** can be propagated only in human 293 cells that supply the deleted E1 gene functions in trans. Unfortunately, the use of high titers of E1-deleted **vectors** has been repeatedly demonstrated to result in low-level expression of viral genes still resident in the **vector**. In addition, the generation of replication-competent Ad (RCA) by recombination events with the E1 sequences residing in 293 cells further limits the usefulness of E1-deleted Ad **vectors**. We addressed these problems by isolating new Ad **vectors** deleted for the E1, E3, and the E2b gene functions. The new **vectors** can be readily grown to high titers and have several improvements, including an increased carrying capacity and a theoretically decreased risk for generating RCA. We have also demonstrated that the further block to Ad **vector** replication afforded by the deletion of both the E1 and E2b genes significantly diminished Ad late gene expression in comparison to a conventional E1-deleted **vector**, without destabilization of the modified **vector** genome. The results suggested that these modified **vectors** may be very useful both for in vitro and in vivo gene therapy applications.

ACCESSION NUMBER: 83307850 MEDLINE
DOCUMENT NUMBER: 83307850 PubMed ID: 6615164
TITLE: Adenoviruses and human tumors: regulation of eukaryotic chromatin structure?.
AUTHOR: Berencsi G; Nasz I
SOURCE: ARCHIV FUR GESCHWULSTFORSCHUNG, (1983) 53 (3) 239-52. Journal code: 746; 0372411. ISSN: 0003-911X.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198310
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19831021

AB Adenoviruses possess four early gene clusters. **E1A**, and **E1B** sequences are directly involved in the initiation of transformation of both rodent, and human cells. Oncogenic potential of the in vitro immortalized rodent cells is also dependent on the expression of **E1A**, and **E1B**. Genes **E2A**, and **E2B** are coding for DNA-binding proteins. These regions may influence the frequency of in vitro transformation if complete virions were used for the immortalization of rodent cells. The existence of the E3 gene cluster, which has been shown to be non-essential to the productive replication in certain host cells, is unique for the adenovirus family. Foreign DNA fragments inserted into E3 or replacing it may modify the host range of adenovirus, and may initiate oncogenic transformation of rodent cells. May cellular oncogenes replace the non-essential region of the adenoviral genome? Are host-range modifications, the formation of defective genomes, and interactions of viral, and cellular DNA in vivo biological properties of adenoviruses? These questions are discussed in the light of recent findings concerning virus-coded functions which may modify chromatin structure, and may be associated with oncogenic potential of adenoviruses in the natural hosts.

L21 ANSWER 12 OF 33 MEDLINE
ACCESSION NUMBER: 1998285701 MEDLINE
DOCUMENT NUMBER: 98285701 PubMed ID: 9621003
TITLE: Characterization of **wild-type** adeno-associated virus type 2-like particles generated during **recombinant** viral vector production and strategies for their elimination.
AUTHOR: Wang X S; Khuntirat B; Qing K; Ponnazhagan S; Kube D M; Zhou S; Dwarki V J; Srivastava A
CORPORATE SOURCE: Department of Microbiology and Immunology, Indiana University School of Medicine, Indianapolis, Indiana 46202, USA.
CONTRACT NUMBER: HL-48342 (NHLBI)
HL-53586 (NHLBI)
HL-58881 (NHLBI)
+
SOURCE: JOURNAL OF VIROLOGY, (1998 Jul) 72 (7) 5472-80.
Journal code: KCV; 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199807
ENTRY DATE: Entered STN: 19980713
Last Updated on STN: 19980713
Entered Medline: 19980701

AB The pSub201-pAAV/Ad plasmid cotransfection system was developed to eliminate homologous **recombination** which leads to generation of the **wild-type** (wt) adeno-associated virus type 2 (AAV) during **recombinant** vector production. The extent of contamination with wt AAV has been documented to range between 0.01 and 10%. However, the precise mechanism of generation of the contaminating wt AAV remains unclear. To characterize the wt AAV genomes, **recombinant** viral stocks were used to infect human **293** cells in the presence of **adenovirus**. Southern blot analyses of viral replicative DNA intermediates revealed that the contaminating AAV genomes were not authentic wt but rather wt AAV-like sequences derived from **recombination** between (i) AAV inverted terminal repeats (ITRs) in the **recombinant** plasmid and (ii) AAV sequences in the helper plasmid. Replicative AAV DNA fragments, isolated following amplification through four successive rounds of amplification in **adenovirus**-infected **293** cells, were molecularly cloned and subjected to nucleotide sequencing to identify the **recombinant** junctions. Following sequence analyses of 31 different ends of AAV-like genomes derived from two different **recombinant** vector stocks, we observed that all **recombination** events involved 10 nucleotides in the AAV D sequence distal to viral hairpin structures. We have recently documented that the first 10 nucleotides in the D sequence proximal to the AAV hairpin structures are essential for successful replication and encapsidation of the viral genome (X.-S. Wang et al., J. Virol. 71:3077-3082, 1997), and it was noteworthy that in each **recombinant** junction sequenced, the same 10 nucleotides were retained. We also observed that **adenovirus** ITRs in the helper plasmid were involved in illegitimate **recombination** with AAV ITRs, deletions of which significantly reduced the extent of wt AAV-like particles. Furthermore, the combined use of **recombinant** AAV plasmids lacking the distal 10 nucleotides in the D sequence and helper

L18 ANSWER 11 OF 11 MEDLINE
ACCESSION NUMBER: 88275039 MEDLINE
DOCUMENT NUMBER: 88275039 PubMed ID: 3292790
TITLE: High-level eucaryotic in vivo expression of biologically active measles virus hemagglutinin by using an adenovirus type 5 helper-free **vector** system.
AUTHOR: Alkhatib G; Briedis D J
CORPORATE SOURCE: Department of Microbiology and Immunology, McGill University, Montreal, Quebec, Canada.
SOURCE: JOURNAL OF VIROLOGY, (1988 Aug) 62 (8) 2718-27.
Journal code: KCV; 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198808
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880819

AB The entire measles virus (MV) hemagglutinin (HA)-coding region was reconstructed from cloned cDNAs and used as part of a hybrid transcription unit to replace a region of the adenovirus type 5 genome corresponding to the entire E1a transcription unit and most of the E1b transcription unit. The resulting recombinant virus was stable and able to replicate to high titers in 293 cells (which constitutively express the complementary **E1a-E1b** functions) in the absence of helper virus. During infection of 293 cells, the hybrid virus expressed MV HA protein which was indistinguishable from that expressed in MV-infected cells in terms of immunoreactivity, gel mobility, glycosylation, subcellular localization, and biologic activity. Infection of 293 cells with the hybrid virus led to high-level synthesis of the MV HA protein (equivalent to 65 to 130% of the level seen in MV-infected cells). At late times after high-multiplicity hybrid virus infection of HeLa and Vero cells (which do not express E1 functions), the level of HA protein synthesis was at least 35% of that seen in 293 cells. This MV-adenovirus recombinant will be useful in the study of the biologic properties of the MV HA protein and in assessment of the potential usefulness of hybrid adenoviruses as live-virus vaccine **vectors**.